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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/648,310	08/25/2000	Paul B. Fisher	62943/JPW/JML	6406

7590 01/30/2003

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EXAMINER	
YU, MISOOK	

ART UNIT	PAPER NUMBER
1642	17

DATE MAILED: 01/30/2003

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/648,310	FISHER ET AL.	
	Examiner	Art Unit	
	MISOOK YU, Ph.D.	1642	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *21 May 2001 and 28 June 2002* .

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☐ Claim(s) *1,9,10,13-22,26,30,34,36,40 and 44-53* is/are pending in the application.

4a) Of the above claim(s) *13-22,26,34,36 and 40* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1,9,10,30 and 44-53* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. §§ 119 and 120

13)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449) Paper No(s) _____ .
4) ☐ Interview Summary (PTO-413) Paper No(s). _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☒ Other: *Seq. alignment* .

The Examiner of your application in the USPTO has changed. To aid in correlating any papers for this application, all further correspondence regarding this application should be directed to Examine Misook Yu.

DETAILED ACTION

Election/Restrictions

Applicant's election with traverse of group I corresponding claims 1, 9, 10, and 30 in Paper No. 6 is acknowledged. The traversal is on the ground(s) that group III invention drawn to the protein encoded by the elected invention i.e., nucleic acid molecule is related to the elected invention. This is not found persuasive because protein and nucleic acid are two different products for the reasons set forth in the previous Office Action (Paper No. 5).

The requirement is still deemed proper and is therefore made FINAL.

Claims 1, 9, 10, 13-22, 26, 30, 34, 36, 40, and 44-53 are pending. Applicant is reminded that applicant instructed the Office to cancel claims 4, 6, 8, and 25 in the preliminary amendment, therefore applicant's instruction in the amendment (Paper No. 10) received on 7-08-2002 to amend claims 4, 6, 8, and 25 cannot be entered because the claims do not exist.

Claims 13-22, 26, 34, 36, and 40 are withdrawn from further consideration pursuant to 37 CFR 1.142(b), as being drawn to a nonelected invention, there being no allowable generic or linking claim. Applicant timely traversed the restriction (election) requirement in Paper No. 6.

Claims 1, 9, 10, and 30 and the new claims 44-53 are examined to the extent they are drawn to nucleic acid molecule.

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 9, 10, 30, 46, 48, and 50 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

Claims 46, 48, and 50 all recite "the polynucleotide sequence shown in SEQ ID NO:2" but it is not clear what the metes and bounds are for the limitation. SEQ ID NO:2 is polypeptide sequence. For the purpose of this Office Action, this examiner will assume that Claims 46, 48, and 50 are drawn to an nucleic acid molecule encoding SEQ ID NO:2. However, this treatment does not relieve applicant the burden of responding to this rejection.

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 51 and 52 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. The claims are drawn to various tumor cells as host cells containing an nucleic acid encoding an PSGen 13. The specification lists the several cancer cells to be treated with instant invention at page 7 lines 5-25. However, the support for the new claims 51 and 52 i.e., various tumor cells to host the instant nucleic acid is not apparent to the examiner. Applicant is requested to point out the support for claims 51 and 52 in the originally filed specification.

Claim 53 is rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention. Claim 53 is drawn to a pharmaceutical composition comprising a nucleic acid encoding PSGen 13. Inherent in a pharmaceutical composition is in vivo use. Since the instant specification mostly talks about cancer

treatment (note for example, page 7 of the specification), the claim is interpreted as a pharmaceutical for cancer treatment. The specification teaches at Fig. 6, 8 teaches that PSGen suppresses phenotype of highly oncogenic cell lines and also teach PSGen suppresses certain promoter activity at Fig. 9 and 10. The *in vitro* suppression of tumoriogenic phenotype cannot be correlated to the invention as claimed, because the *in vitro* assay the protein is in contact with target cells and are not subjected to the defense of the body. In addition, characteristics of cultured cell lines generally differ significantly from the characteristics of in vivo primary cancers or metastatic cancers. Freshney (Culture of Animal Cells, A Manual of Basic Technique, Alan R. Liss, Inc., 1983, New York, page 4) teach that it is recognized in the art that there are many differences between cultured cells and their counterparts *in vivo*. These differences stem from the dissociation of cells from a three-dimensional geometry and their propagation on a two-dimensional substrate. Specific cell interactions characteristic of histology of the tissue are lost. The culture environment lacks the input of the nervous and endocrine systems involved in homeostatic regulation *in vivo*. Without this control, cellular metabolism may be more constant *in vitro* but may not be truly representative of the tissue from which the cells were derived. This has often led to tissue culture being regarded in a rather skeptical light (p. 4, see Major Differences *In Vitro*). Further, Dermer (Bio/Technology, 1994, 12:320) teaches that, "petri dish cancer" is a poor representation of malignancy, with characteristics profoundly different from the human disease. Further, Dermer teaches that when a normal or malignant body cell adapts to immortal life in culture, it takes an evolutionary -type step that enables the new line to thrive in its artificial environment. This step transforms a cell from one that is stable and differentiated to one that is not, yet normal or malignant cells *in vivo* are not like that. The reference states that evidence of the contradictions between life on the bottom of a lab dish and in the body has been in the scientific literature for more than 30 years. Clearly it is well known in the art that cells in culture exhibit characteristics different from those *in vivo* and cannot duplicate the complex conditions of the *in vivo* environment involved in host-tumor and cell-cell interactions. Thus, based on the cell culture data presented in the specification, it could not be predicted that the instant invention could

kill tumor cells *in vivo*. In addition, anti-tumor agents and those that prevent, reduce, retard or eliminate secretion of metastatic promoters, must accomplish several tasks to be effective. They must be delivered into the circulation that supplies the tumor or metastatic promotor producing cells and interact at the proper site of action and must do so at a sufficient concentration and for a sufficient period of time. It is clear, as disclosed above that the specification does not teach how to make/use a formulation with a targeting molecule. Also, the target cell must not have an alternate means of survival despite action at the proper site for the drug. In addition variables such as biological stability, half-life or clearance from the blood are important parameters in achieving successful therapy. The formulation may be inactivated *in vivo* before producing a sufficient effect, for example, by degradation, immunological activation or due to an inherently short half life of the formulation. One cannot extrapolate the teachings of the specification to the claim because it is well known that the art of anticancer drug discovery for cancer therapy is highly unpredictable, for example, Gura (Science, 1997, 278:1041-1042) teaches that researchers face the problem of sifting through potential anticancer agents to find ones promising enough to make human clinical trials worthwhile and teach that since formal screening began in 1955, many thousands of drugs have shown activity in either cell or animal models but that only 39 have actually been shown to be useful for chemotherapy (p. 1041, see first and second para of column 1). Because of the known unpredictability of the art, in the absence of experimental evidence, no one skilled in the art would accept the assertion that the claimed peptide would be useful for treating cancer. Further, the refractory nature of cancer to drugs is well known in the art. Jain (Sci. Am., 1994, 271:58-65) teaches that tumors resist penetration by drugs (p.58, col 1) and that scientists need to put expanded effort into uncovering the reasons why therapeutic agents that show encouraging promise in the laboratory often turn out to be ineffective in the treatment of common solid tumors (p. 65, col 3). Curti (Crit. Rev. in Oncology/Hematology, 1993, 14:29-39) teaches that solid tumors resist destruction by chemotherapy agents and that although strategies to overcome defense mechanisms of neoplastic cells have been developed and tested in a number of patients, success has been limited and further teaches that it

is certainly possible that cancer cells possess many as yet undefined additional molecular mechanisms to defeat chemotherapy treatment strategies and if this is true, designing effective chemotherapeutic regimens for solid tumors may prove a daunting task (para bridging pages 29-30) and concludes that knowledge about the physical barriers to drug delivery in tumors is a work in progress (p. 36, col 2). It is clear that based on the state of the art, in the absence of experimental evidence, no one skilled in the art would accept the assertion that the claimed peptide would be useful for treating cancer. In addition, Hartwell et al (Science, 1997, 278:1064-1068) teach that an effective chemotherapeutic must selectively kill tumor cells, that most anticancer drugs have been discovered by serendipity and that the molecular alterations that provide selective tumor cell killing are unknown and that even understanding the detailed molecular mechanism by which a drug acts often provides little insight into why the treated tumor cell dies (para bridging pages 1064-1065) and Jain (cited supra) specifically teaches that systemic treatment typically consists of chemotherapeutic drugs that are toxic to dividing cells (p. 58, col 2, para 2).

Art recognize cancer treatment is not a trivial matter. The specification provides insufficient guidance with regard to these issues and provides no working examples which would provide guidance to one skilled in the art and no evidence has been provided which would allow one of skill in the art to predict the efficacy of the claimed invention.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 1, 30, 44, 46, 48, and 53 are rejected under 35 U.S.C. 102(b) as being anticipated by GenBank accession number AA891725 (08-Jan-1999).

Claims 1, 30, 44, 46, 48, and 53 are interpreted as drawn to an isolated nucleic acid per se encoding a PSGen 13 protein.

GenBank accession number AA891725 teaches an isolated DNA molecule encoding a rat protein which is identical to instant SEQ ID NO:2. Note the sequence alignment. Also compare instant Fig.1 showing cDNA encoding rat PSGen13 with GenBank accession number AA891725.

Claims 1, 9, 10, 30, 44, and 53 are rejected under 35 U.S.C. 102(a) as being anticipated by Fisher (WO 99/43844, 02-Sept-1999).

Claims 1, 9, 10, 30, 44, and 53 are interpreted as drawn to an isolated nucleic acid per se encoding a PSGen 13 protein, vector, and host cells.

Fisher (WO 99/43844) teaches an isolated nucleic acid PSGen at Fig. 35B, claim 21, vector and host cell at page 47.

Conclusion

SEQ ID NO:1 is free of art.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to MISOOK YU, Ph.D. whose telephone number is 703-308-2454. The examiner can normally be reached on 8 A.M. to 5:30 P.M., every other Friday off.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Anthony C Caputa can be reached on 703-308-3995. The fax phone numbers for the organization where this application or proceeding is assigned are 703-305-3014 for regular communications and 703-872-9307 for After Final communications.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to the receptionist whose telephone number is 703-308-0196.

Misook Yu

January 19, 2003

ANTHONY C. CAPUTA
SUPERVISORY PATENT EXAMINER
TECHNOLOGY CENTER 1600

XX
DR WPI; 2002-280914/32.
DR P-PSDB; AAU76532.
XX
PT New rat and human Progression Suppressed Gene 13 for preventing the
PT growth of cancer cells and/or new blood vessels, and for treating
PT patients suffering from a cancer -
XX
PS Claim 2; Fig 1; 53pp; English.
XX
CC The invention relates to novel isolated nucleic acids which encode a
CC rat or human Progression Suppressed Gene 13 (PSGen 13) protein. The
CC nucleic acids are useful for preventing the growth of cancer cells and/or
CC new blood vessels, and for treating patients suffering from a cancer,
CC e.g. nasopharyngeal tumour, thyroid tumour, leukaemia, lymphoma, or
CC cancer of the breast, lung, prostate, ovary or colon. PSGen 13 may also
CC be used to suppress the transformed phenotype of a malignant cell.
CC Administration of PSGen 13 gene or protein may result in a decrease in
tumour mass, number of cancer cells, serum tumour marker, tumour
metastasis, vascularisation, perfusion, or rate of tumour growth,
improved clinical symptoms, and/or increased patient survival. The
CC present sequence represents the coding sequence of rat Progression
CC Suppressed Gene 13 (rPSGen 13).
XX
SQ Sequence 780 BP; 223 A; 151 C; 187 G; 219 T; 0 other;

Query Match 100.0%; Score 780; DB 24; Length 780;
Best Local Similarity 100.0%; Pred. No. 1e-209;
Matches 780; Conservative 0; Mismatches 0; Indels 0; Gaps 0;

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Qy      1 GGCACGAGCTCTCCTCGTCCCCTCCCTTCTCCACTGCAGCCTTTCTCTTAGCCCGAACCA 60
          ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db      1 GGCACGAGCTCTCCTCGTCCCCTCCCTTCTCCACTGCAGCCTTTCTCTTAGCCCGAACCA 60

Qy     61 CTTCCTTCTTCTGCTTGTTCCTCCCTAGGGCGCGGAAGCTGAGTGCAGGGTTCAGACCCA 120
          ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db     61 CTTCCTTCTTCTGCTTGTTCCTCCCTAGGGCGCGGAAGCTGAGTGCAGGGTTCAGACCCA 120

Qy    121 CGCGGCGAGCAGCTCTTCAGTGAAGAAGGAAGCAATCGGAGGGTCAGCAATGAACGTGGA 180
          ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db    121 CGCGGCGAGCAGCTCTTCAGTGAAGAAGGAAGCAATCGGAGGGTCAGCAATGAACGTGGA 180

Qy    181 GCATGAGGTTAACCTCCTGGTGGAGGAAATTCATCGTCTGGGTTCCAAAAATGCCGATGG 240
          ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db    181 GCATGAGGTTAACCTCCTGGTGGAGGAAATTCATCGTCTGGGTTCCAAAAATGCCGATGG 240

Qy    241 GAAACTGAGTGTGAAGTTTGGGGTCCTCTTCCAAGACGACAGATGTGCCAATCTCTTTGA 300
          ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db    241 GAAACTGAGTGTGAAGTTTGGGGTCCTCTTCCAAGACGACAGATGTGCCAATCTCTTTGA 300

Qy    301 AGCGTTGGTGGGAACTCTGAAAGCCGCAAAACGAAGGAAGATTGTTACGTACGCAGGAGA 360
          ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db    301 AGCGTTGGTGGGAACTCTGAAAGCCGCAAAACGAAGGAAGATTGTTACGTACGCAGGAGA 360

Qy    361 GCTGCTTTTGCAAGGTGTTCATGATGATGTTGACATTGTATTGCTGCAAGATTAATGTGG 420
          ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db    361 GCTGCTTTTGCAAGGTGTTCATGATGATGTTGACATTGTATTGCTGCAAGATTAATGTGG 420

Qy    421 TTTGCAGATCTGGGGGTATCTGGTAAACTGGAATAATTAAGTTAAAGGACAAACATGAAG 480
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Qy    481 TTCCTTATGTATTTTTATAGACCTTTGTAAACAAAAGGGGACTTGTTGAGAAGTCCTGTT 540
          ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db    481 TTCCTTATGTATTTTTATAGACCTTTGTAAACAAAAGGGGACTTGTTGAGAAGTCCTGTT 540

Qy    541 TTTATACCTTGGAGCAAAACATTACAATGTAAAAATAAACAAAACCTGTTATTTTTTTTT 600
          ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
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Qy    601 TCTTAAGAAGGTAATCGGGAGACGTAGGCAATAAAATGTTTTCAGAGGTGCGAAAAAGCT 660
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Qy    661 TTTGTTTTCTTAAACCATTCTTAGTCTCTGCCACACTTGACACTCCGTCAAAGTGAGAAG 720
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Db    661 TTTGTTTTCTTAAACCATTCTTAGTCTCTGCCACACTTGACACTCCGTCAAAGTGAGAAG 720

Qy    721 CGAACTAAAGACCAACTGCGGTGGAAAATATTATGTTTATGTAATAAAAAAAAATCATGT 780
          ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db    721 CGAACTAAAGACCAACTGCGGTGGAAAATATTATGTTTATGTAATAAAAAAAAATCATGT 780
```

RESULT 2
AAZ21517
ID AAZ21517 standard; cDNA; 800 BP.
XX
AC AAZ21517;
XX
DT 01-DEC-1999 (first entry)
XX
DE Progression suppressed gene 13 (PSGen13).
XX
KW Progression suppressed gene; PSGen; progression elevated gene; PEGen;
KW tumour; reciprocal subtraction differential RNA display; RSDD;
KW differential expression; gene cloning; cancer; ss.
XX
OS Rattus sp.
XX
FH Key Location/Qualifiers
FT CDS 170..448
FT /*tag= a
FT /product= PSGen13
FT /note= "Progression suppressed gene 13 protein"
XX
PN WO9943844-A1.
XX
PD 02-SEP-1999.
XX
PF 26-FEB-1999; 99WO-US04323.
XX
PR 27-FEB-1998; 98US-0032684.
PR 03-NOV-1998; 98US-0185115.
PR 23-NOV-1998; 98US-0197889.
XX
PA (UYCO) UNIV COLUMBIA NEW YORK.
XX
PI Fisher PB;
XX
DR WPI; 1999-550872/46.
DR P-PSDB; AAY39325.
XX
PT Identifying nucleic acids differentially expressed between two samples,
PT particularly sequences involved in tumour progression -
XX
PS Claim 21; Fig 35B; 110pp; English.
XX
CC This sequence is the progression suppressed gene 13 (PSGen13). This gene
CC has suppressed expression in progressed tumour cells. PSGen13 was
CC identified using new methods for identifying nucleic acids differentially
CC expressed between two samples. The method involves performing reciprocal
CC subtraction differential RNA display (RSDD) between the two samples to
CC generate two subtraction samples. The subtraction samples are amplified
CC and compared to identify those nucleic acids that are differentially
CC expressed. The method is used to identify and clone differentially
CC expressed genes, particularly those with increased or reduced expression
CC during tumour cell progression, e.g. progression suppressed genes (PSGen)
CC and progression elevated genes (PEGen). The method reduces the complexity
CC of the band pattern produced in conventional differential RNA display
CC (where bands may be obscured, resulting in false positive signals) since
CC most bands common to both samples are eliminated, allowing identification
CC and cloning of genes displaying anticipated differential expression. RSDD
CC requires only a single anchored primer for amplification and reamplified
CC cDNA can be analysed by reverse Northern blotting.
XX
SQ Sequence 800 BP; 243 A; 153 C; 185 G; 219 T; 0 other;

```
  Query Match             96.8%;  Score 754.8;  DB 20;  Length 800;
  Best Local Similarity   99.5%;  Pred. No. 1.3e-202;
  Matches  778;  Conservative    0;  Mismatches    2;  Indels    2;  Gaps    2;

Qy       1 GGCACGAGCTCTCCTCGTCCCCTCCCTTCTCCACTGCAGCCTTTCTCTTAGCCCGAACCA 60
           ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db       1 GGCACGAGCTCTCCTCGTCCCCTCCCTTCTCCACTGCAGCCTTTCTCTTAGCCCGAACCA 60

Qy      61 CTTCCTTCTTCTGCTTGTTCCTCCCTAGGGCGCGGAAGCTGAGTGCAGGGTTCAGACCCA 120
           ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db      61 CTTCCTTCTTCTGCTTGTTCCTCCCTAGGGCGCGGAAGCTGAGTGCAGGGTTCAGACCCA 120

Qy     121 CGCGGCGAGCAGCTCTTCAGTGAAGAAGGAAGCAATCGGAGGGTCAGCAATGAACGTGGA 180
           ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db     121 CGCGGCGAGCAGCTCTTCAGTGAAGAAGGAAGCAATCGGAGGGTCAGCAATGAACGTGGA 180

Qy     181 GCATGAGGTTAACCTCCTGGTGGAGGAAATTCATCGTCTGGGTTCCAAAAATGCCGATGG 240
           ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db     181 GCATGAGGTTAACCTCCTGGTGGAGGAAATTCATCGTCTGGGTTCCAAAAATGCCGATGG 240

Qy     241 GAAACTGAGTGTGAAGTTTGGGGTCCTCTTCCAAGACGACAGATGTGCCAATCTCTTTG- 299
           ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||| 
Db     241 GAAACTGAGTGTGAAGTTTGGGGTCCTCTTCCAAGACGACAGATGTGCCAATCTCTTTGA 300

Qy     300 AAGCGTTGGTGGGAACTCTGAAAG-CCGCAAAACGAAGGAAGATTGTTACGTACGCAGGA 358
           || |||||||||||||||||||| |||||||||||||||||||||||||||||||||| |
Db     301 AACCGTTGGTGGGAACTCTGAAAGCCCGCAAAACGAAGGAAGATTGTTACGTACGCAGAA 360

Qy     359 GAGCTGCTTTTGCAAGGTGTTCATGATGATGTTGACATTGTATTGCTGCAAGATTAATGT 418
           ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db     361 GAGCTGCTTTTGCAAGGTGTTCATGATGATGTTGACATTGTATTGCTGCAAGATTAATGT 420

Qy     419 GGTTTGCAGATCTGGGGGTATCTGGTAAACTGGAATAATTAAGTTAAAGGACAAACATGA 478
           ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db     421 GGTTTGCAGATCTGGGGGTATCTGGTAAACTGGAATAATTAAGTTAAAGGACAAACATGA 480

Qy     479 AGTTCCTTATGTATTTTTATAGACCTTTGTAAACAAAAGGGGACTTGTTGAGAAGTCCTG 538
           ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db     481 AGTTCCTTATGTATTTTTATAGACCTTTGTAAACAAAAGGGGACTTGTTGAGAAGTCCTG 540

Qy     539 TTTTTATACCTTGGAGCAAAACATTACAATGTAAAAATAAACAAAACCTGTTATTTTTTT 598
           ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db     541 TTTTTATACCTTGGAGCAAAACATTACAATGTAAAAATAAACAAAACCTGTTATTTTTTT 600

Qy     599 TTTCTTAAGAAGGTAATCGGGAGACGTAGGCAATAAAATGTTTTCAGAGGTGCGAAAAAG 658
           ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db     601 TTTCTTAAGAAGGTAATCGGGAGACGTAGGCAATAAAATGTTTTCAGAGGTGCGAAAAAG 660

Qy     659 CTTTTGTTTTCTTAAACCATTCTTAGTCTCTGCCACACTTGACACTCCGTCAAAGTGAGA 718
           ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db     661 CTTTTGTTTTCTTAAACCATTCTTAGTCTCTGCCACACTTGACACTCCGTCAAAGTGAGA 720

Qy     719 AGCGAACTAAAGACCAACTGCGGTGGAAAATATTATGTTTATGTAATAAAAAAAAATCAT 778
           ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db     721 AGCGAACTAAAGACCAACTGCGGTGGAAAATATTATGTTTATGTAATAAAAAAAAATCAT 780

Qy     779 GT 780
           ||
Db     781 GT 782
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RESULT 3
ABK11086
ID   ABK11086 standard; cDNA; 835 BP.
XX
AC   ABK11086;
XX
DT   05-JUN-2002  (first entry)
XX
DE   Human Progression Suppressed Gene 13 (HuPSGen 13), cDNA.
XX
KW   Human; Progression Suppressed Gene 13; rPSGen 13; cytostatic; cancer;
KW   blood vessel; nasopharyngeal tumour; thyroid tumour; leukaemia;
KW   lymphoma; breast; lung; prostate; ovary; colon; gene; ss.
XX
OS   Homo sapiens.
XX
FH   Key             Location/Qualifiers
FT   CDS             197..442
FT                   /*tag= a
FT                   /product= "Progression suppressed gene 13 protein"
XX
PN   WO200216419-A2.
XX
PD   28-FEB-2002.
XX
PF   27-AUG-2001; 2001WO-US26795.
XX
PR   25-AUG-2000; 2000US-0648310.
XX
PA   (UYCO ) UNIV COLUMBIA NEW YORK.
XX
PI   Fisher PB,  Kang D,  Su Z;
XX
DR   WPI; 2002-280914/32.
DR   P-PSDB; AAU76533.
XX
PT   New rat and human Progression Suppressed Gene 13 for preventing the
PT   growth of cancer cells and/or new blood vessels, and for treating
PT   patients suffering from a cancer
XX
PS   Claim 4; Fig 2; 53pp; English.
XX
CC   The invention relates to novel isolated nucleic acids which encode a
CC   rat or human Progression Suppressed Gene 13 (PSGen 13) protein. The
CC   nucleic acids are useful for preventing the growth of cancer cells and/or
CC   new blood vessels, and for treating patients suffering from a cancer,
CC   e.g. nasopharyngeal tumour, thyroid tumour, leukaemia, lymphoma, or
CC   cancer of the breast, lung, prostate, ovary or colon. PSGen 13 may also
CC   be used to suppress the transformed phenotype of a malignant cell.
CC   Administration of PSGen 13 gene or protein may result in a decrease in
CC   tumour mass, number of cancer cells, serum tumour marker, tumour
CC   metastasis, vascularisation, perfusion, or rate of tumour growth,
CC   improved clinical symptoms, and/or increased patient survival. The
CC   present sequence represents the coding sequence of human Progression
CC   Suppressed Gene 13 (HuPSGen 13).
XX
SQ   Sequence 835 BP; 246 A; 160 C; 176 G; 253 T; 0 other;
```

```
  Query Match             43.0%;  Score 335.2;  DB 24;  Length 835;
  Best Local Similarity   69.1%;  Pred. No. 2.9e-84;
  Matches  539;  Conservative    0;  Mismatches  218;  Indels   23;  Gaps    5;

Qy      16 CGTCCCCTCCCTTCTCCACTGCAGCCTTTCTCTTAGCCCGAACCACTTCCTTCTTCTGCT 75
           | | | |  ||| |||||| || | |||||||| |||  | |||||||||| |  ||
Db      43 CCTGCTCCTCCTCCTCCACAGCCGTCTTTCTCTTTGCCTCAGCCACTTCCTTCCTTCGCC 102

Qy      76 TGTTCCTCCCTAGGGCGCGGAAGCTGAGTGCAGGGTTCAGACCCACGCGGCGAGCAGCTC 135
           |   |||||| || || | ||||  |    | |||| ||||||||||||||||
Db     103 TCACCCTCCCCAGTGCACTGAAGAAGGTAACCGGGTCCAGACCCACGCGGCGCCAGTTCT 162

Qy     136 TTCAGTGAAGAAGGAAGCAATCGGAGGGTCAGCAATGAACGTGGAGCATGAGGTTAACCT 195
           | |     || |   | ||| | |||||||||| ||||| || ||||||||||
Db     163 CCGGCGGGAAGGAAAACCGCGCAGAGAGGCAGCAATGAATGTGGATCACGAGGTTAACCT 222

Qy     196 CCTGGTGGAGGAAATTCATCGTCTGGGTTCCAAAAATGCCGATGGGAAACTGAGTGTGAA 255
           | | |||||||||||||||||| ||||||| |||||||| ||||| ||  | || |||||
Db     223 CTTAGTGGAGGAAATTCATCGTTTGGGTTCAAAAAATGCTGATGGAAAGTTAAGCGTGAA 282

Qy     256 GTTTGGGGTCCTCTTCCAAGACGACAGATGTGCCAATCTCTTTGAAGCGTTGGTGGGAAC 315
           |||||||||||||||  || || | |||||||| |||||||||| ||||| |||||
Db     283 ATTTGGGGTCCTCTTCCGTGATGATGATAAATGTGCCAACCTCTTTGAAGCATTGGTAGGAAC 342

Qy     316 TCTGAAAGCCGCAAAACGAAGGAAGATTGTTACGTACGCAGGAGAGCTGCTTTTGCAAGG 375
           ||| ||||| |||||||||||||||||||| || ||  |||||||||||||| |||||||
```

tail. The sequence tag present in the cDNA between the NotI site and the oligo-dT track served to verify it as a clone from the normalized osteoblast library cDNA Library Preparation: M.B. Soares Lab Clone distribution: clones will be available through Research Genetics (www.resgen.com)
Seq primer: M13 Forward
POLYA=Yes.

```
FEATURES             Location/Qualifiers
     source          1. .690
                     /organism="Rattus norvegicus"
                     /strain="Sprague-Dawley"
                     /db_xref="taxon:10116"
                     /clone="UI-R-DR1-ckz-m-14-0-UI"
                     /clone_lib="UI-R-DR1"
                     /dev_stage="adult"
                     /lab_host="DH10B (Life Technologies)"
                     /note="Vector: pT7T3D-Pac (Pharmacia) with a modified
                     polylinker; Site_1: Not I; Site_2: Eco RI; The UI-R-DR1
                     library is a normalized Rat Osteoblast library (nREO)
                     constructed in pT3T7 vector according to the procedure
                     described by Bonaldo, Lennon & Soares (Normalization and
                     Subtraction:  Two Approaches to Facilitate Gene Discovery.
                     Genome Research 6:  791-806, 1996).  The oligonucleotide
                     used to prime first strand synthesis contained the
                     sequence tag AAGATATCAA between the Not I cloning site and
                     dT18 stretch. The Rat Osteoblast tissue was provided by
                     Lian & Stein of the University of Massachussetts Medical
                     School.
                     TAG_LIB=UI-R-DR1
                     TAG_TISSUE=osteoblast
                     TAG_SEQ=AAGATATCAA"
BASE COUNT      188 a    163 c    109 g    230 t
ORIGIN
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Query Match 82.8%; Score 646; DB 14; Length 690;
Best Local Similarity 99.0%; Pred. No. 4.7e-152;
Matches 661; Conservative 0; Mismatches 5; Indels 2; Gaps 1;

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Qy    113 CAGACCCACGCGGCGAGCAGCTCTTCAGTGAAGAAGGAAGCAATCGGAGGGTCAGCAATG 172
Db    690 CAGACCCACGCGGCGAGCAGCTCTTCAGTGAAGAAGGAAGCAATCGGAGGGTCAGCAATG 631

Qy    173 AACGTGGAGCATGAGGTTAACCTCCTGGTGGAGGAAATTCATCGTCTGGGTTCCAAAAAT 232
Db    630 AACGTGGAGCATGAGGTTAACCTCCTGGTGGAGGAAATTCATCGTCTGGGTTCCAAAAAT 571

Qy    233 GCCGATGGGAAACTGAGTGTGAAGTTTGGGGTCCTCTTCCAAGACGACAGATGTGCCAAT 292
Db    570 GCCGATGGGAAACTGAGTGT--AAGTTTGGGGCCTCTTCCAAGACGACAGATGTGCCAAT 513

Qy    293 CTCTTTGAAGCGTTGGTGGGAACTCTGAAAGCCGCAAAACGAAGGAAGATTGTTACGTAC 352
Db    512 CTCTTTGAAGCGTTGGTGGGAACTCTGAAAGCCGCAAAACGAAGGAAGATTGTTACGTAC 453

Qy    353 GCAGGAGAGCTGCTTTTGCAAGGTGTTCATGATGATGTTGACATTGTATTGCTGCAAGAT 412
Db    452 GCAGGAGAGCTGCTTTTGCAAGGTGTTCATGATGATGTTGACATTGTATTGCTGCAAGAT 393

Qy    413 TAATGTGGTTTGCAGATCTGGGGGTATCTGGTAAACTGGAATAATTAAGTTAAAGGACAA 472
Db    392 TAATGTGGTTTGCAGATCTGGGGGTATTTGGTAAACTGGAATAATTAAGTTAAAGGACAA 333

Qy    473 ACATGAAGTTCCTTATGTATTTTTATAGACCTTTGTAAACAAAAGGGGACTTGTTGAGAA 532
Db    332 ACATGAAGTTCCTTATGTATTTTTATAGACCTTTGTAAACAAAAGGGGACTTGTTGAGAA 273

Qy    533 GTCCTGTTTTTATACCTTGGAGCAAAACATTACAATGTAAAAATAAACAAAACCTGTTAT 592
Db    272 GTCCTGTTTTTATACCTTGGAGCAAAACATTACAATGTAAAAATAAACAAAACCTGTTAT 213

Qy    593 TTTTTTTTTCTTAAGAAGGTAATCGGGAGACGTAGGCAATAAAATGTTTTCAGAGGTGCG 652
Db    212 TTTTTTTTTCTTAAGAAGGTAATCGGGAGACGTAGGCAATAAAATGTTTTCAGAGGTGCG 153

Qy    653 AAAAAGCTTTTGTTTTCTTAAACCATTCTTAGTCTCTGCCACACTTGACACTCCGTCAAA 712
Db    152 AAAAAGCTTTTGTTTTCTTAAACCATTCTTAGTCTCTGCCACACTTGACACTCCGTCAAA 93

Qy    713 GTGAGAAGCGAACTAAAGACCAACTGCGGTGGAAAATATTATGTTTATGTAATAAAAAAA 772
Db     92 GTGAGAAGCGAACTAAAGACCAACTGCGGTGGAAAATATTATGTTTATGTAATAAAAAAA 33

Qy    773 AATCATGT 780
Db     32 AATCATGT 25
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```
RESULT 2
AA891725/c
LOCUS       AA891725                642 bp    mRNA    linear   EST 08-JAN-1999
DEFINITION  EST195528 Normalized rat kidney, Bento Soares Rattus sp. cDNA clone
            RKIAG02 3' end, mRNA sequence.
ACCESSION   AA891725
VERSION     AA891725.1  GI:4132080
KEYWORDS    EST.
SOURCE      Rattus sp.
  ORGANISM  Rattus sp.
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Rodentia; Sciurognathi; Muridae; Murinae;
            Rattus.
REFERENCE   1  (bases 1 to 642)
  AUTHORS   Lee,N.H., Glodek,A., Chandra,I., Mason,T.M., Quackenbush,J.,
            Kerlavage,A.R. and Adams,M.D.
  TITLE     Rat Genome Project: Generation of a Rat EST (REST) Catalog & Rat
            Gene Index
  JOURNAL   Unpublished (1998)
COMMENT     On Apr 3, 1998 this sequence version replaced gi:3018604.
            Contact: Lee, NH
            The Institute for Genomic Research
            9712, Medical Center Drive, Rockville, MD 20850, USA
            Tel: (301)-838-3529
            Fax: (301)-838-0208
            Email: nhlee@tigr.org
            Seq primer: M13-21.
FEATURES             Location/Qualifiers
     source          1. .642
                     /organism="Rattus sp."
                     /db_xref="taxon:10118"
                     /clone="RKIAG02"
                     /clone_lib="Normalized rat kidney, Bento Soares"
                     /note="Organ: kidney; Vector: pT7T3Pac; Site_1: EcoRI;
                     Site_2: NotI"
BASE COUNT      184 a    155 c     99 g    204 t
ORIGIN
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Query Match 81.5%; Score 636; DB 9; Length 642;
Best Local Similarity 100.0%; Pred. No. 1.6e-149;
Matches 636; Conservative 0; Mismatches 0; Indels 0; Gaps 0;

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Qy    145 GAAGGAAGCAATCGGAGGGTCAGCAATGAACGTGGAGCATGAGGTTAACCTCCTGGTGGA 204
Db    642 GAAGGAAGCAATCGGAGGGTCAGCAATGAACGTGGAGCATGAGGTTAACCTCCTGGTGGA 583

Qy    205 GGAAATTCATCGTCTGGGTTCCAAAAATGCCGATGGGAAACTGAGTGTGAAGTTTGGGGT 264
Db    582 GGAAATTCATCGTCTGGGTTCCAAAAATGCCGATGGGAAACTGAGTGTGAAGTTTGGGGT 523

Qy    265 CCTCTTCCAAGACGACAGATGTGCCAATCTCTTTGAAGCGTTGGTGGGAACTCTGAAAGC 324
Db    522 CCTCTTCCAAGACGACAGATGTGCCAATCTCTTTGAAGCGTTGGTGGGAACTCTGAAAGC 463

Qy    325 CGCAAAACGAAGGAAGATTGTTACGTACGCAGGAGAGCTGCTTTTGCAAGGTGTTCATGA 384
Db    462 CGCAAAACGAAGGAAGATTGTTACGTACGCAGGAGAGCTGCTTTTGCAAGGTGTTCATGA 403

Qy    385 TGATGTTGACATTGTATTGCTGCAAGATTAATGTGGTTTGCAGATCTGGGGGTATCTGGT 444
```

Start codon

Stop codon

```
                ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db    402 TGATGTTGACATTGTATTGCTGCAAGATTAATGTGGTTTGCAGATCTGGGGGTATCTGGT 343

Qy    445 AAACTGGAATAATTAAGTTAAAGGACAAACATGAAGTTCCTTATGTATTTTTATAGACCT 504
                ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db    342 AAACTGGAATAATTAAGTTAAAGGACAAACATGAAGTTCCTTATGTATTTTTATAGACCT 283

Qy    505 TTGTAAACAAAAGGGGACTTGTTGAGAAGTCCTGTTTTTATACCTTGGAGCAAAACATTA 564
                ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db    282 TTGTAAACAAAAGGGGACTTGTTGAGAAGTCCTGTTTTTATACCTTGGAGCAAAACATTA 223

Qy    565 CAATGTAAAAATAAACAAAACCTGTTATTTTTTTTTTCTTAAGAAGGTAATCGGGAGACG 624
                ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db    222 CAATGTAAAAATAAACAAAACCTGTTATTTTTTTTTTCTTAAGAAGGTAATCGGGAGACG 163

Qy    625 TAGGCAATAAAATGTTTTCAGAGGTGCGAAAAAGCTTTTGTTTTCTTAAACCATTCTTAG 684
                ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db    162 TAGGCAATAAAATGTTTTCAGAGGTGCGAAAAAGCTTTTGTTTTCTTAAACCATTCTTAG 103

Qy    685 TCTCTGCCACACTTGACACTCCGTCAAAGTGAGAAGCGAACTAAAGACCAACTGCGGTGG 744
                ||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||||
Db    102 TCTCTGCCACACTTGACACTCCGTCAAAGTGAGAAGCGAACTAAAGACCAACTGCGGTGG 43

Qy    745 AAAATATTATGTTTATGTAATAAAAAAAAATCATGT 780
                ||||||||||||||||||||||||||||||||||
Db     42 AAAATATTATGTTTATGTAATAAAAAAAAATCATGT 7
```

RESULT 3
AK013984

```
LOCUS       AK013984                769 bp    mRNA    linear   HTC 19-JAN-2002
DEFINITION  Mus musculus 13 days embryo head cDNA, RIKEN full-length enriched
            library, clone:3110003A17:homolog to PRO2013, full insert sequence.
ACCESSION   AK013984
VERSION     AK013984.1  GI:12851586
KEYWORDS    HTC; CAP trapper.
SOURCE      Mus musculus (strain:C57BL/6J) 13 days embryo head cDNA to mRNA,
            clone_lib:RIKEN full-length enriched mouse cDNA library
            clone:3110003A17.
  ORGANISM  Mus musculus
            Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi;
            Mammalia; Eutheria; Rodentia; Sciurognathi; Muridae; Murinae; Mus.
REFERENCE   1
  AUTHORS   Carninci,P. and Hayashizaki,Y.
  TITLE     High-efficiency full-length cDNA cloning
  JOURNAL   Meth. Enzymol. 303, 19-44 (1999)
  MEDLINE   99279253
   PUBMED   10349636
REFERENCE   2
  AUTHORS   Carninci,P., Shibata,Y., Hayatsu,N., Sugahara,Y., Shibata,K.,
            Itoh,M., Konno,H., Okazaki,Y., Muramatsu,M. and Hayashizaki,Y.
  TITLE     Normalization and subtraction of cap-trapper-selected cDNAs to
            prepare full-length cDNA libraries for rapid discovery of new genes
  JOURNAL   Genome Res. 10 (10), 1617-1630 (2000)
  MEDLINE   20499374
   PUBMED   11042159
REFERENCE   3
  AUTHORS   Shibata,K., Itoh,M., Aizawa,K., Nagaoka,S., Sasaki,N., Carninci,P.,
            Konno,H., Akiyama,J., Nishi,K., Kitsunai,T., Tashiro,H., Itoh,M.,
            Sumi,N., Ishii,Y., Nakamura,S., Hazama,M., Nishine,T., Harada,A.,
            Yamamoto,R., Matsumoto,H., Sakaguchi,S., Ikegami,T., Kashiwagi,K.,
            Fujiwake,S., Inoue,K., Togawa,Y., Izawa,M., Ohara,E., Watahiki,M.,
            Yoneda,Y., Ishikawa,T., Ozawa,K., Tanaka,T., Matsuura,S., Kawai,J.,
            Okazaki,Y., Muramatsu,M., Inoue,Y., Kira,A. and Hayashizaki,Y.
  TITLE     RIKEN integrated sequence analysis (RISA) system--384-format
            sequencing pipeline with 384 multicapillary sequencer
  JOURNAL   Genome Res. 10 (11), 1757-1771 (2000)
  MEDLINE   20530913
   PUBMED   11076861
REFERENCE   4
  AUTHORS   Kawai,J., Shinagawa,A., Shibata,K., Yoshino,M., Itoh,M., Ishii,Y.,
            Arakawa,T., Hara,A., Fukunishi,Y., Konno,H., Adachi,J., Fukuda,S.,
            Aizawa,K., Izawa,M., Nishi,K., Kiyosawa,H., Kondo,S., Yamanaka,I.,
            Saito,T., Okazaki,Y., Gojobori,T., Bono,H., Kasukawa,T., Saito,R.,
            Kadota,K., Matsuda,H., Ashburner,M., Batalov,S., Casavant,T.,
            Fleischmann,W., Gaasterland,T., Gissi,C., King,B., Kochiwa,H.,
            Kuehl,P., Lewis,S., Matsuo,Y., Nikaido,I., Pesole,G.,
            Quackenbush,J., Schriml,L.M., Staubli,F., Suzuki,R., Tomita,M.,
            Wagner,L., Washio,T., Sakai,K., Okido,T., Furuno,M., Aono,H.,
            Baldarelli,R., Barsh,G., Blake,J., Boffelli,D., Bojunga,N.,
            Carninci,P., de Bonaldo,M.F., Brownstein,M.J., Bult,C.,
            Fletcher,C., Fujita,M., Gariboldi,M., Gustincich,S., Hill,D.,
            Hofmann,M., Hume,D.A., Kamiya,M., Lee,N.H., Lyons,P.,
            Marchionni,L., Mashima,J., Mazzarelli,J., Mombaerts,P., Nordone,P.,
            Ring,B., Ringwald,M., Rodriguez,I., Sakamoto,N., Sasaki,H.,
            Sato,K., Schonbach,C., Seya,T., Shibata,Y., Storch,K.F., Suzuki,H.,
            Toyo-oka,K., Wang,K.H., Weitz,C., Whittaker,C., Wilming,L.,
            Wynshaw-Boris,A., Yoshida,K., Hasegawa,Y., Kawaji,H., Kohtsuki,S.
            and Hayashizaki,Y.
  TITLE     Functional annotation of a full-length mouse cDNA collection
  JOURNAL   Nature 409 (6821), 685-690 (2001)
  MEDLINE   21085660
   PUBMED   11217851
REFERENCE   5  (bases 1 to 769)
  AUTHORS   Adachi,J., Aizawa,K., Akahira,S., Akimura,T., Aono,H., Arai,A.,
            Arakawa,T., Baldarelli,R., Bono,H., Brownstein,M., Bult,C.,
            Carninci,P., Fukuda,S., Fukunishi,Y., Furuno,M., Hanagaki,T.,
            Hara,A., Hayatsu,N., Hill,D., Hiramoto,K., Hiraoka,T., Hori,F.,
            Hume,D., Imotani,K., Ishii,Y., Itoh,M., Izawa,M., Kasukawa,T.,
            Kato,H., Kawai,J., Kojima,Y., Konno,H., Kouda,M., Koya,S.,
            Kurihara,C., Matsuyama,T., Miyazaki,A., Nishi,K., Nomura,K.,
            Numazaki,R., Ohno,M., Okazaki,Y., Okido,T., Owa,C., Quackenbush,J.,
            Saito,H., Saito,R., Sakai,C., Sakai,K., Sano,H., Sasaki,D.,
            Schriml,L., Shibata,K., Shibata,Y., Shinagawa,A., Shiraki,T.,
            Sogabe,Y., Suzuki,H., Tagami,M., Tagawa,A., Takahashi,F.,
            Tanaka,T., Tejima,Y., Toya,T., Yamamura,T., Yamanaka,I.,
            Yasunishi,A., Yoshida,K., Yoshino,M., Muramatsu,M. and
            Hayashizaki,Y.
  TITLE     Direct Submission
  JOURNAL   Submitted (10-JUL-2000) Yoshihide Hayashizaki, The Institute of
            Physical and Chemical Research (RIKEN), Laboratory for Genome
            Exploration Research Group, RIKEN Gemomic Sciences Center (GSC),
            RIKEN Yokohama Institute; 1-7-22 Suehiro-cho, Tsurumi-ku, Yokohama,
            Kanagawa 230-0045, Japan (E-mail:genome-res@gsc.riken.go.jp,
            URL:http://genome.gsc.riken.go.jp/, Tel:81-45-503-9222,
            Fax:81-45-503-9216)
COMMENT     Please visit our web site (http://genome.gsc.riken.go.jp/) for
            further details.
            cDNA library was prepared and sequenced in Mouse Genome
            Encyclopedia Project of Genome Exploration Research Group in Riken
            Genomic Sciences Center and Genome Science Laboratory in RIKEN.
            Division of Experimental Animal Research in Riken contributed to
            prepare mouse tissues. First strand cDNA was primed with a primer
            [5' GAGAGAGAGAAGGATCCAAGAGCTCTTTTTTTTTTTTTTTVN 3'], cDNA was
            prepared by using trehalose thermo-activated reverse transcriptase
            and subsequently enriched for full-length by cap-trapper. cDNA went
            through one round of normalization to Rot = 10.0 and subtraction to
            Rot = 50.0. Second strand cDNA was prepared with the primer adapter
            of sequence [5'
            GAGAGAGAGATTCTCGAGTTAATTAAATTAATCCCCCCCCCCCCCC 3']. cDNA was cleaved
            with XhoI and SstI. Cloning sites, 5' end: XhoI; 3' end: SstI.
            Host: SOLR.
FEATURES                Location/Qualifiers
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                        /organism="Mus musculus"
                        /strain="C57BL/6J"
                        /db_xref="FANTOM_DB:3110003A17"
                        /db_xref="MGD:MGI:1906054"
                        /db_xref="taxon:10090"
                        /clone="3110003A17"
                        /tissue_type="head"
                        /clone_lib="RIKEN full-length enriched mouse cDNA library"
                        /dev_stage="13 days embryo"
     CDS                155. .400
                        /note="data source:SPTR, source key:Q9P1F3, evidence:ISS
                        homolog to PRO2013
```